Journal of Cardiology Cases (2011) 4, e1—e4

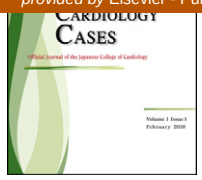
View metadata, citation and similar papers at core.ac.uk

brought to you by CORE

provided by Elsevier - Publisher Connector

ScienceDirect

journal homepage: www.elsevier.com/locate/jccase



Case Report

Acute myocardial infarction involving double vessel total occlusion of the left anterior descending and left circumflex arteries: A case report

Hidehira Fukaya (MD, PhD)[a,b,*], Jun Oikawa (MD)[a,b], Shoji Hirasawa (MD, PhD)[a,b], Takao Shimohama (MD, PhD)[b], Taiki Tojo (MD, PhD)[b], Shinichi Niwano (MD, PhD)[b], Tohru Izumi (MD, PhD, FJCC)[b]

[a] *Division of Cardiology, Shizuoka Municipal Shimizu Hospital, Shizuoka, Japan*
[b] *Department of Cardio-Angiology, School of Medicine, Kitasato University, Sagamihara, Japan*

Received 7 January 2011; received in revised form 8 April 2011; accepted 11 May 2011

KEYWORDS
Acute myocardial infarction;
Intra-aortic balloon pump;
Double vessel occlusion

Summary A 66-year-old Japanese man complained of chest pain consistent with acute myocardial infarction (AMI). His electrocardiogram showed ST segment elevation in the anterior and inferior leads. Emergency coronary angiography revealed occlusion of the proximal left anterior descending artery (LAD) and middle left circumflex artery (LCx). An intra-aortic balloon pump (IABP) was inserted to restore antegrade coronary flow in these vessels. Coronary stents were subsequently implanted at the culprit lesions. Although previous reports of multivessel coinstantaneous AMI are rare and indicate a poor prognosis, he had a relatively benign course and was discharged with New York Heart Association functional class I without post-operational complications.
© 2011 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.

Introduction

Multivessel simultaneous occlusion of coronary arteries is a rare condition with a poor prognosis [1]. Here, we present a case of acute myocardial infarction (AMI) with simultaneous total occlusion of the left anterior descending artery (LAD) and left circumflex artery (LCx). Inconsistent with previous reports [1—3], this patient was discharged without symptoms or subsequent complications.

Case report

A 66-year-old Japanese man visited his local clinic complaining of left anterior chest pain of sudden onset. His risk factors for coronary artery disease were hypertension, diabetes mellitus, dyslipidemia, habitual smoking (20

* Corresponding author at: 1-15-1 Kitasato, Minami-ku, Sagamihara Kanagawa 252-0374, Japan. Tel.: +81 42 778 8111; fax: +81 42 778 9983.
E-mail address: hidehira@med.kitasato-u.ac.jp (H. Fukaya).

1878-5409/$ — see front matter © 2011 Japanese College of Cardiology. Published by Elsevier Ltd. All rights reserved.
doi:10.1016/j.jccase.2011.05.002

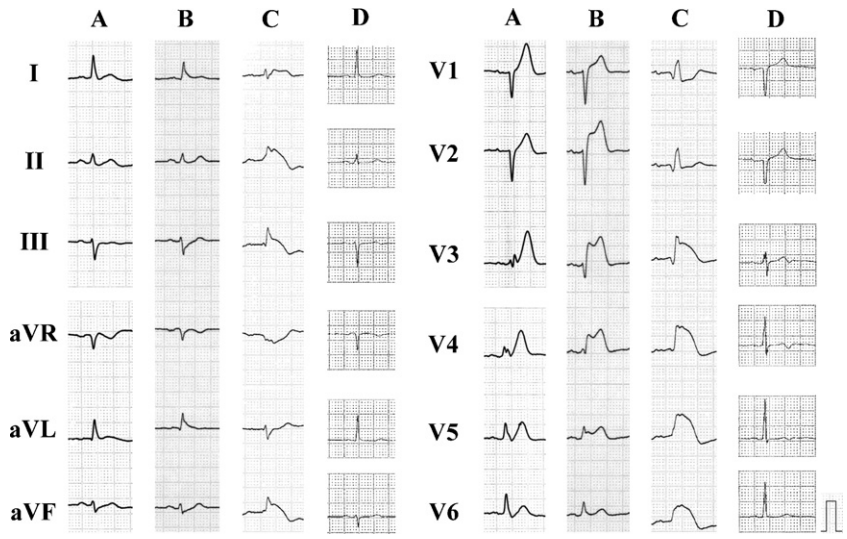


Figure 1 Changes in ECG over time. ECGs were obtained at the patient's local clinic (A), our ED (B), our ED just before CAG (C), and after 4 months (D). (A) Tall T wave in anterior lead. (B) ST segment elevation in anterior lead. (C) Intraventricular conduction delay and additional ST segment elevation in inferior lead. (D) Poor R progression and left axis deviation. ECG, electrocardiogram; ED, emergency department; CAG, coronary angiography.

cigarettes/day for the last 50 years), and past cerebral lacunar infarction. The 12-lead electrocardiogram (ECG) showed a tall T wave in V1—V5 (Fig. 1A), indicating anterior AMI. He was immediately referred to our hospital's emergency department (ED), where his blood pressure (161/95 mmHg) and pulse (90 beat per min) were found to be relatively stable. One hour after his reported chest pain, the ECG showed ST segment elevation in the anterior leads (Fig. 1B). The blood test at admission exhibited renal insufficiency but did not show the abnormal elevation of creatinine phosphokinase (CPK), which is indicative of myocardial injury. Serum creatinine level was 1.63 mg/dL, calculated as 34.2 ml/min/1.73 m^2 by the estimated glomerular filtration rate. The echocardiograph showed hypokinetic wall motion at the anteroseptal area. Other areas showed no distinct abnormality, but also no hyperkinesis indicative of a compensative effect. Left ventricular ejection fraction (LVEF) was calculated as 50% by the 2-dimensional Simpson's method. After about 30 min, his ECG results were exacerbated and his symptoms worsened. Just before coronary angiography (CAG), the ECG revealed intraventricular conduction delay and additional ST segment elevation in the inferior leads (Fig. 1C). Aspirin 200 mg was given by oral and 3000 U low-molecular heparin by intravenous administration before CAG.

Emergency CAG was performed using the right femoral approach, revealing total occlusion of the proximal LAD and middle LCx (Fig. 2A and B). There was no collateral flow from the right coronary artery to the LAD or LCx. ECG at this time showed the same findings as in Fig. 1C, indicating that this ECG change apparently resulted from double vessel occlusion. During the angiography, the patient's blood pressure fell to 70/30 mmHg, necessitating the insertion of an intra-aortic balloon pump (IABP) through a sheath introducer placed in the right femoral artery. By this IABP support, the development of delayed antegrade coronary flow in both coronary arteries was presented, and then the hemodynamic condition was recovered. ECG subsequently showed a gradual recovery in ST segment elevation, particularly in the inferior leads, and recovery of QRS width to normal. Another sheath introducer was inserted at the right brachial artery because of tortuous change in the left external iliac artery. At first, a 6 Fr left Judkins guiding catheter (JL3.5) was set at the ostium of the left coronary artery, and a 0.014″ guidewire (Runthrough NS®, Terumo, Tokyo, Japan) was passed through to the LAD. Subsequently, a 2.0 mm × 15 mm angioplasty balloon (Lacrosse®, Goodman, Nagoya, Japan) was inserted at the culprit lesion and inflated (6 atm), allowing undelayed antegrade flow. A 3.0 mm × 15 mm drug-eluting stent (DES; Xience V®, Abbott, Tokyo, Japan) was then implanted (8 atm) and correct stent positioning was confirmed using intravascular ultrasonography (IVUS). The wire was then was crossed to the LCx, and another 0.014″ guidewire (Sion®, ASAHI INTECC CO., LTD., Nagoya, Japan) was crossed to the side branch because the culprit was a bifurcation lesion. The balloon was inflated in the bilateral branches, and a 2.5 mm × 18 mm DES (Xience V) was implanted at the culprit lesion. IVUS was performed, and additional inflation by the non-compliant balloon (Powered Lacrosse®, Goodman, Nagoya, Japan) was performed to ensure suitable attachment of the DES to the intima. We did not perform the kissing-balloon technique for this bifurcated lesion because the antegrade flow in the posterolateral branch was not disturbed and a large dosage of contrast media would induce the deterioration of renal insufficiency. Finally, Thrombolysis In Myocardial Infarction (TIMI) grade III flow was observed in both the LAD and the LCx (Fig. 2C and D). We performed IVUS in both coronary arteries after pre-dilatation by small-sized balloon, and coronary stenting. The IVUS findings after ballooning (Fig. 3) showed the lipid-rich, soft plaque and significant stenosis in both culprit lesions.

After the procedure, a Swan-Ganz catheter was inserted and the initial hemodynamic parameter was categorized

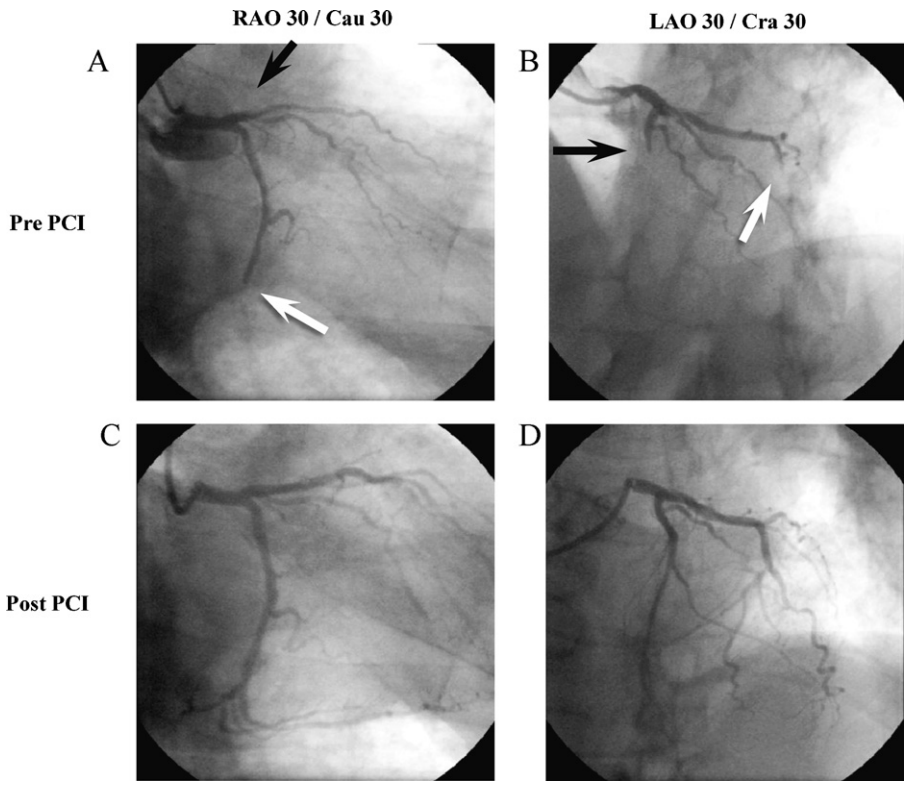


Figure 2 CAG results. CAG results in RAO-caudal view (A) and LAO-cranial view (B). Total occlusion of the proximal LAD (black arrow) and middle LCx (white arrow) are visible. The final CAG after PCI is also shown in same direction (C) RAO-caudal and (D) LAD-cranial. TIMI grade III flow was observed in both the LAD and the LCx. CAG, coronary angiography; LCx, left circumflex artery; LAD, left anterior descending artery; RAO, right anterior oblique; LAO, left anterior oblique; PCI, percutaneous coronary intervention; TIMI, Thrombolysis In Myocardial Infarction.

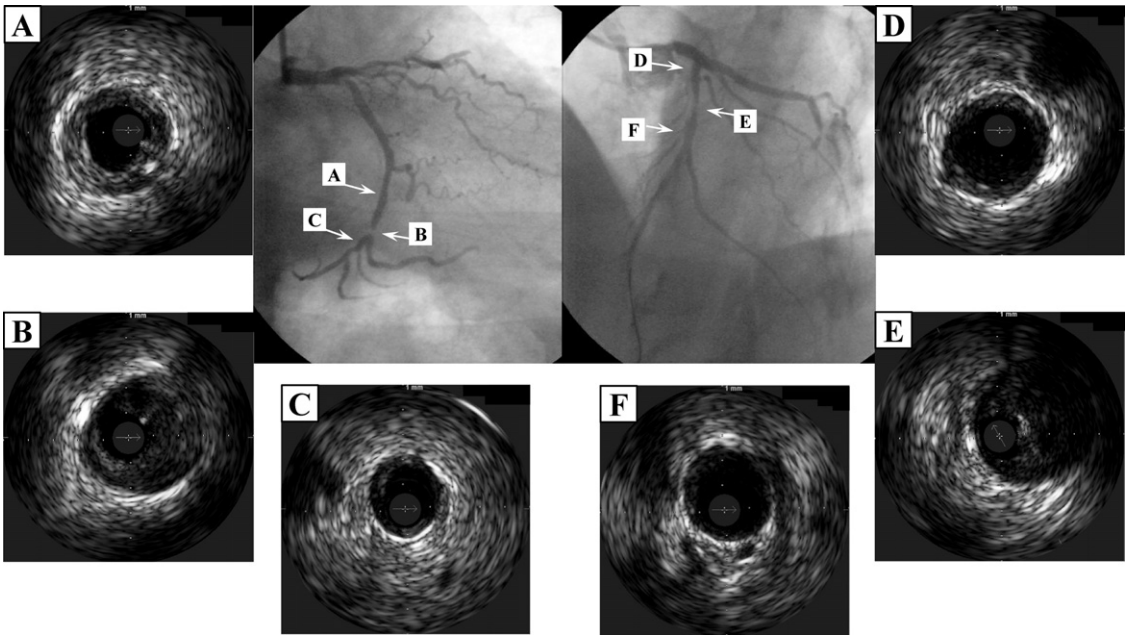


Figure 3 IVUS results. Left and right panels show IVUS results for the LCx and LAD, respectively. (A, B, and C) Show the area proximal to the culprit, the culprit, and distal to it in the LCx, respectively. (D), (E), and (F) Show the same respective areas in the LAD. Both coronary artery lesions contained lipid-rich plaque. In particular, (E) shows findings of attenuation, indicating lipid-rich, vulnerable plaque. IVUS, intravascular ultrasound; LAD, left anterior descending artery; LCx, left circumflex artery.

under subset IV of the Forrester classification. Although the patient experienced transient renal malfunction, his overall clinical course after admission was relatively benign. Serum CPK level peaked at 3744 IU/L. By day 2, his hemodynamic parameters were stabilized and the IABP was removed. Renal function had also recovered to that at admission. At day 18, he was discharged without symptoms or subsequent complications. Fig. 1D shows the ECG after 4 months, which indicates poor R progression but no abnormal Q waves in the inferior leads. Echocardiography performed at the same time showed slight hypokinesis in the anteroseptal area only, and LVEF was kept at 60%. As at writing he has experienced no complications.

Discussion

AMI commonly occurs through occlusion of a coronary artery by atheromatous plaque rupture followed by thrombus formation [4]. In the present case, these phenomena occurred simultaneously in two coronary vessels. Acute occlusion of two or three coronary vessels is extremely rare, and shows poor prognosis and high mortality. One study reported that multiple coronary thrombi were observed in about 10% of postmortem cases of acute coronary syndrome (ACS) [5]. The development of multiple coronary occlusions is often fatal, resulting in sudden cardiac death, and thus accurate diagnosis of multivessel coronary occlusion is rarely achieved in clinical settings. Another group reported that among their non-ST elevated ACS patients, 79% had at least one plaque rupture outside of the culprit lesion; 70.8% had at least one rupture in an artery other than the culprit artery; and 12.5% had at least one rupture in all three arteries [6]. These authors suggested that although multiple coronary artery occlusions are rare, ACS patients may always be at risk of this life-threatening condition [6].

Further, even if patients with multivessel AMI are admitted before reaching a fatal or unstable state, the hospital mortality rate is still high because of reduced cardiac function. In the present patient, the changes noted in his ECG over time suggest the possibility that occlusion of the LAD was followed by that of LCx, delaying the collapse of his hemodynamics. The IVUS findings indicated the presence of lipid-rich plaque in both culprit lesions. Given that blood tests showed no hematological or coagulation disorders, these results indicate that the double vessel occlusion resulted from local plaque rupture, although IVUS showed no clear findings to differentiate between ruptured plaque or ulceration. Comparing these findings, the LAD culprit contained more lipid-rich plaque, showing attenuation. These findings suggest that this patient would have been better treated with thrombectomy and distal protection.

The patient underwent IABP, which was first introduced in 1970s and has since been reported as effective [7—9]. IABP is recommended by the guidelines of the Japanese Circulation Society for management of patients with ST-elevation myocardial infarction [10]. As a device, IABP is relatively easy to handle and thus allows for rapid insertion. In the present study, minimizing the time required to stabilize the hemodynamics and recanalize the occluded vessels appears to have contributed to the patient's discharge without subsequent complications. The ECG after 4 months showed recovery of the R wave in precordal leads, although it also showed poor R progression. Further, no Q wave abnormality was seen in the inferior leads. Echocardiography at that time also indicated almost normal contraction in the inferior area and the maintenance of LVEF. We consider that IABP in this patient contributed to the prevention of a large myocardial infarction, and to stabilization of the patient's hemodynamics in the early phase.

References

[1] Sia SK, Huang CN, Ueng KC, Wu YL, Chan KC. Double vessel acute myocardial infarction showing simultaneous total occlusion of left anterior descending artery and right coronary artery. Circ J 2008;72:1034—6.
[2] Hosokawa S, Hiasa Y, Miyamoto H, Suzuki N, Takahashi T, Kishi K, Tanimoto M, Ohtani R. Acute myocardial infarction showing total occlusion of right coronary artery and thrombus formation of left anterior descending artery. Jpn Heart J 2001;42:365—9.
[3] Boztosun B, Gurel E, Gunes Y, Olcay A. Myocardial infarction associated with thrombus formation in non-culprit coronary arteries. J Thromb Thrombolysis 2007;23:73—5.
[4] Goldstein JA, Demetriou D, Crines CL, Pica M, Shoukfeh M, O'Neill WW. Multiple complex coronary plaques in patients with acute myocardial infarction. New Eng J Med 2000;343:915—22.
[5] Arbustini E, Dal Bello B, Morbini P, Burke AP, Bocciarelli M, Specchia G, Virmani R. Plaque erosion is a major substrate for coronary thrombosis in acute myocardial infarction. Heart 1999;82:269—72.
[6] Rioufol G, Finet G, Ginon I, André-Fouët X, Rossi R, Vialle E, Desjoyaux E, Convert G, Huret JF, Tabib A. Multiple atherosclerotic plaque rupture in acute coronary syndrome: a three-vessel intravascular ultrasound study. Circulation 2002;106:804—8.
[7] Thiele H, Sick P, Boudriot E, Diederich KW, Hambrecht R, Niebauer J, Schuler G. Randomized comparison of intra-aortic balloon support with a percutaneous left ventricular assist device in patients with revascularized acute myocardial infarction complicated by cardiogenic shock. Eur Heart J 2005;26:1276—83.
[8] Ohman EM, Hochman JS. Aortic conterpulsation in acute myocardial infarction: physiologically important but does the patient benefit? Am Heart J 2001;141:889—92.
[9] Barron HV, Every NR, Parsons LS, Angeja B, Goldberg RJ, Gore JM, Chou TM. Investigators in the National Registry of Myocardial Infarction 2. The use of intra-aortic balloon counterpulsation in patients with cardiogenic shock complication acute myocardial infarction: data from the National Registry of Myocardial Infarction 2. Am Heart J 2001;141:933—9.
[10] Takano T, Ogawa A, Kasanuki H, Kimura K, Goto Y, Sumiyoshi T, Shirota H, Tanaka K, Nagao K, Hirayama A, Makuuchi H, Yamaguchi T, Yamashina A, Yoshino H, Asano R, et al. Guideline for the management of patients with ST-elevation myocardial infarction (JCS 2008). Circ J 2008;72(Suppl. IV): 1347—442.